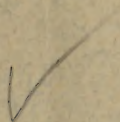


Burnett (S. M.)

INDEX
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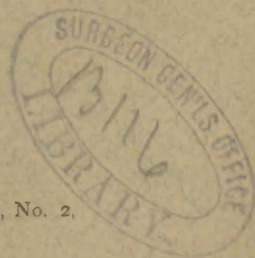
A CASE OF ACUTE CHEMOSIS

BY



SWAN M. BURNETT, M.D.

Lecturer on Ophthalmology and Otology in the Medical Department of Georgetown University, Surgeon-in-charge of the Ophthalmic Division Central Dispensary, and one of the Consulting Ophthalmic and Aural Surgeons to Providence Hospital, Washington, D. C.



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IN the June number of the *Klin. Monatsbl. f. Augenheilk.* for 1870, Dr. Zehender relates a case of what he calls "acutes Bindehautödem," the history of which is briefly as follows:

The patient was a man of 55 years, and a convalescent from an attack of left-sided pneumonia.

On April 6th, in the evening, he was seized with symptoms of a severe catarrh, accompanied by headache. The next day a considerable chemosis was noted, which increased until the conjunctiva extruded like a bladder from between the lids. On the 10th it had reached its maximum. On the 12th the œdema had entirely disappeared from the left, and on the 14th from the right eye, leaving a marked conjunctival injection.

The only treatment was atropine and rest in bed. There was no heart trouble, and no signs of pyæmia.

As I can find no account of any similar cases in the literature at my command, I judge them to be rare, and as they are interesting on account of the obscurity of their causation, I am led to record a case that fell under my observation a year ago, which bears a great similarity to that of Zehender just related.

Mr. G. D. M., a young lawyer of 29, small of stature and of a nervous temperament, had been studying with undue vigor for months past, working, he states, continuously about 20 hours out of the 24.

Four weeks previously he had a throat trouble, terminating in abscess, which was followed by an inflammation of the right eye. Some "smokiness" in the vision of this eye caused him to consult me. $V = \frac{4}{8}$. Externally the eye was normal, but with the ophthalmoscope all around the periphery of the choroid of *both* eyes, atrophic spots were seen, and in the vicinity of the macula of the *R.E.* there was an apparent thinning of that membrane and a few fine points of pigment. The V.F. was perfect, and both eyes were emmetropic.

Ordered smoked glasses and complete rest. As there was also an evident malarial complication, he was ordered good doses of quinia.

On April 29th, three days after I first saw him, I was called to see him at his house, he being unable to leave his room. I learned that on the previous evening, at 9 o'clock, he had been seized with an intolerable pain in the *left* eye and left side of the head, which had persisted throughout the entire night. He had nausea and rigors which were apparently nervous in their character, and were not followed by any notable elevation in temperature. The eye was exceedingly sensitive to light, but there was no very considerable injection of the conjunctiva and the pupil responded promptly to light. I ordered opiates, hot applications, and a continuation of the quinia. On the morning of the 30th I found the lids somewhat red and swollen, and a marked chemosis which was almost perfectly clear. The photophobia was the same, but the pain was less. On May 1st the condition was unaltered, but on my visit on the morning of the 2d I found the photophobia almost entirely gone and the chemosis greatly diminished. No pain whatever, appetite returning, which before had been lost. Vision was not then tested, but on the 5th, when he visited me at my office, I found it $\frac{4}{8}$. There was a very slight conjunctival injection, and the ophthalmoscope showed a slight haziness of the vitreous. In the course of a week both eyes had $V = \frac{4}{8}$.

He now says that the attack in the other (right) eye some weeks ago was very similar in its character to this.

The causes usually in operation for the production of the chemotic condition are obstructions of some character to

the return of blood through the veins, such as inflammations of the uveal tract and of the conjunctiva itself. These are what Shies * calls *active* or inflammatory causes, in contradistinction to those causes which are unattended with signs of inflammation and are called *passive*, because the fluid exudes passively through the walls of the vessels as in ordinary cases of œdema from relaxation of the vascular walls.

In the case just related we have no substantial evidence that either one of these causes was in predominant operation. That there was not an inflammation of any part of the uveal tract of sufficient violence to cause obstruction of the venous circulation, is abundantly shown by the fact of the abrupt termination of the symptoms, and the want of objective evidence. There was some turbidity of the vitreous it is true, but that I account for by a condition of the interior blood-vessels similar to that of the outer coats of the eye which allowed transudation of fluid into that cavity.

It is more than probable, however, that the pathological condition of the ant. portion of the choroid played the part of predisposing cause, for evidently, judging from the ophthalmoscopic appearances, morbid changes were slowly taking place there. These however, were not in and of themselves sufficient to cause the circulatory obstruction necessary to the chemosis. I look upon the neuralgia of the fifth pair (probably of malarial origin), as the exciting cause.

It is well-known that neuralgia of the fifth pair is often attended with a hyperæmia of the conjunctiva. Under ordinary circumstances, this undue determination of blood is carried away by the veins without any serious trouble. In this case, however, owing to the diseased condition of the ant. portion of the choroid, the veins were probably unequal to the task imposed on them and a chemosis was probably the result. At least, I think we are justified in the opinion that the condition was due largely to deranged vaso-motor influence.

* *Klin. Monatsbl. f. Augenheilk.*, 1872, p. 1-7.

